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URINARY DYSFUNCTION IN THE AGED: NEUROLOGICAL CONSIDERATIONS

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THE most frequent expression of urinary dysfunction among the aged is incontinence. This is a common condition and anyone acquainted with rehabilitation facilities, nursing homes, or continuing care units knows its scope.[1] Commonly, the disorder is treated expediently by a Foley catheter. In some cases, the catheter may be a lesser evil than the incontinence, but in others it certainly is not, and the treatment may become more of a problem than the disease. Even if catheterization is tolerated with equanimity, the logistics and expense involved are not insignificant. Catheters must be irrigated; they become obstructed and kinked, get pulled out, and their balloons deflate. Patients with catheters become febrile or septic and develop periurethral abscesses. Physicians must be found to change catheters. In short, chronic catheterization is not an ideal method of management. It seems reasonable then to make some effort to avoid it, and to do this, we must have some knowledge of the underlying cause of urinary dysfunction.

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The imposition of volitional control over bladder function is unusual in that a completely reflex activity becomes subject to cerebral control which is, in turn, structured by social circumstance. Conscious control of bladder function appears to be chiefly exerted by a direct cortical influence on the striated or volitional urinary sphincter. Contraction of the external sphincter constricts the urethra but also has an intraspinal inhibitory effect on the detrusor motor neurons. External sphincteric activity is unusual in that it ceases only during defecation and micturition.[2] Tone, or the activity of a number of motor units, persists even during deep sleep when other skeletal musculature is profoundly relaxed. Thus, conscious participation is not essential to maintain sphincter tone. Volitional contraction of the sphincter will interrupt or prevent voiding, but is possible for only brief periods. The sphincter contracts in response to afferent input from the abdominal cavity with an increase in intraabdominal pressure and to pelvic nerve firing generated by an increase in intravesical volume. It contracts in response to coughing, sneezing, straining, and the upright posture.

Afferent pelvic nerve fibers are thought to synapse through an internuncial nerve directly on the pudendal motor cells within the sacral spinal cord but some pelvic nerve afferents also reach to the brain stem whence they project to subcortical and cortical centers. This pathway from the bladder to subcortical and cortical centers is essential to volitional control of a reflex function. Information on the degree of bladder filling and the imminence of a detrusor contraction is a requisite to impose volitional control.[3,4,5]

The detrusor and the external sphincter maintain a reciprocal relation, but this is disturbed in patients with spinal cord lesions between the sacral cord segments and the brain stem. Patients with suprasacral cord lesions have hyperactive sphincteric responses to bladder filling as a direct effect of afferent pelvic nerve excitation impinging through internuncial neurons on the pudendal motor nucleus. However, the external sphincter fails to relax or explodes with activity during a detrusor contraction.[6] In patients with a normal neuraxis, the external sphincter relaxes completely during detrusor activity. Inappropriate sphincter activity during a detrusor contraction in patients with suprasacral spinal cord lesions implies that the control of the relation between the detrusor and the external sphincter requires supraspinal activity. Because lesions superior to the superior colliculi are associated with precipitant, nonvolitional but coordinate mic-

turition, it appears that the brain stem is the area responsible for coordination of bladder and urethral activity.

Specific Types of Dysfunction Associated with Specific Neural Lesions

Interruption of neural activity at various levels has a definite and predictable effect on lower urinary tract function. Section or damage to the peripheral pelvic nerve interferes with conscious control of reflex events involved in lower urinary tract function in a number of ways. Afferent fibers are interrupted distal to the sacral cord and, therefore, afferent activity cannot reach the brain stem, so that there is no input to subserve either coordination of reflex function or volitional control. If the bladder is deprived *only* of its afferent innervation, it becomes flaccid and tolerates filling with equanimity. There is no increase in pressure up to very large volumes. Incontinence, in this circumstance, results from overflow when the bladder is filled to the limits of its distensibility. The bladder is not trabeculated and is easily managed by intermittent catheterization.

Loss of the motor preganglionic nerves to the bladder clinically results in an "autonomous" bladder which preserves its tone. Indeed, intravesical pressure generated by filling ultimately equals intraurethral resistance and urine is forced out the urethra. There is no neural mechanism to open the urethra because this requires pelvic nerve, sacral cord, and probably brain stem activity. Therefore, a kind of arreflexic voiding occurs which is associated with a relatively large residual urine. Upper urinary tract dilatation is common because intravesical storage pressures are elevated and vesicoureteral reflux is common. Peripheral motor denervation is much more difficult to manage than isolated sensory denervation because in the latter case the bladder usually decompensates. The bladder *may* decompensate after a preganglionic motor lesion, but, more commonly, becomes trabeculated and hypertonic, apparently in a compensatory response. The hypertonic response to filling associated with peripheral motor lesions is not well understood. It appears to be due to the development of a hypersensitivity of the postganglionic neuron in the bladder wall, a hypersensitive motor endplate, or both. Small amounts of cholinergic substances are readily available in the vesical ganglia despite preganglionic transection, and a hypertonic bladder will respond to treatment with anticholinergic agents. These reduce bladder storage pressure and improve storage func-

tion. However, because no mechanism for reflex voiding exists, intermittent catheterization must be used to empty the bladder if anticholinergic agents are employed.

Etiology of Motor and Sensory Peripheral Neuropathy

Diabetes mellitus, tabes dorsalis, and alcoholic peripheral neuropathy are associated with afferent denervation of the bladder. In some cases, a flaccid decompensated bladder may also result from prolonged overdistention associated with obstruction of the bladder outlet, as, for example, in patients with prostatism or in women with large cystoceles. Patients with "atonic" or "flaccid" arreflexic vesical dysfunction are best managed by intermittent catheterization. Diabetic patients tolerate chronic Foley catheterization poorly and should not be treated in this manner if any alternative method of therapy is possible.

Radical pelvic surgery, particularly for malignant tumors of the rectum, prostate, or uterus, is often associated with both motor and sensory denervation.[8] Operations of this kind are occasionally also associated with injury to the hypogastric plexus innervating the proximal urethral smooth musculature or the pudendal nerve innervating the external sphincter. Patients with a heavily trabeculated hypertonic bladder and poor voiding with a large residual urine volume should be evaluated for sphincteric competence. Competence of the internal vesical sphincter can be easily assessed with an upright cystogram. If the cystogram shows an open, patulous, nonfunctional bladder outlet, passive continence is lost. These patients may be helped by small doses of alpha adrenergic agents, but these are contraindicated for patients with severe peripheral vascular disease, hypertension, or hyperthyroidism.

Interference with the innervation of the lower urinary tract at a more central level, either within the sacral cord or sacral dural canal, results in detrusor denervation involving both motor and sensory elements and occasionally damage to the neural elements which contribute to the pudendal nerve. The thoracolumbar sympathetic nerves, which give rise to the hypogastric plexus and innervate the urethral smooth musculature, escape the effects of such a lesion. These patients have heavily trabeculated bladders, with large residual urine volumes, high pressure bladder storage dysfunction, and an arreflexic detrusor. Because internal sphincter function is intact, passive continence is readily achieved by intermittent cathe-

terization, provided the hypertonic response to filling is treated by anticholinergic agents. Patients with this kind of injury preserve some vague sensation of bladder distention apparently conveyed by sensory fibers traveling with the hypogastric nerves since these are spared sacral spinal cord or cauda equina injury.

Patients with urinary retention associated with lumbar intervertebral disc syndromes or after major surgery, as, for example, a fractured hip, are special cases, because the evidence indicates that this is not often the result of a direct neural injury, but rather is usually an overdistention injury. Reflex detrusor activity is difficult to elicit despite painful degrees of bladder overdistention with filling. Because bladder overfilling is painful, presumably the afferent pathway is intact. The depression of reflex activity appears due to muscular injury from overdistention.[9] Repeated episodes of bladder overdistention compound the problem, and should be avoided as the patient is treated by a timed voiding program and intermittent catheterization. Usually, reflex detrusor function is regained in such patients after bladder decompression. There is nothing wrong with chronic Foley catheter drainage to achieve this, but it is less desirable than intermittent catheterization which seems to effect a more rapid recovery of bladder function and is associated with a smaller risk of serious infection.

Suprasacral Spinal Lesions

Typically, demyelinating diseases, vascular disease processes, or injuries which interfere with spinal cord function above the sacral cord segments but below the brain stem result in discoordinate bladder and urethral activity. Hyperreflexic bladder responses to filling are common and are associated with discoordinate external sphincter activity or detrusor-sphincter dyssynergia. In patients with complete loss of spinal cord activity at one level, little is to be gained by treatment with anticholinergic agents with respect to continence because the sensation of bladder filling and warning of detrusor events is absent. However, in patients with partial lesions, typical of the demyelinating diseases, it may be possible to depress hyperreflexic detrusor responses and thus gain continence with anticholinergive agents. If frank detrusor-sphincter dyssynergia is present, treatment by an anticholinergic agent will usually precipitate urinary retention. It is preferable to depress the hyperreflexic sphincteric response with small doses of a skeletal muscle relaxant, as, for example, dantroline Na

(Dantrium), baclofan (Lioresol), or diazepam (Valium), and thereafter institute anticholinergic therapy in small doses.

Suprabulbar Lesions

Patients with suprabulbar lesions often have coordinate but hyperreflexic detrusor responses which occur without warning. In these cases, simultaneous measurement of bladder and urethral pressure, combined with fluoroscopic visualization of the bladder and urethra and constant monitoring of the external sphincter electromyogram, demonstrates a bladder which tolerates filling for a time with equanimity until suddenly there is a suppression of external sphincter electromyographic activity, a fall in urethral closing pressure and a sharp rise in detrusor pressure with visible opening of the bladder neck and proximal urethra and urine flow. Only at the onset of detrusor contraction, or occasionally just at the time of urethral urinary flow, is the patient aware of the bladder contraction. At this time, he vigorously contracts the external sphincter in an effort to halt the proceedings. External sphincter electromyographic activity and urethral closing pressure increase and the patient can stop the detrusor contraction. It appears that the motor limb of the volitional pathway for suppression of the detrusor events is intact, but subconscious or unconscious recognition of detrusor reflex events is lacking. Because the lesion is supraspinal, it appears that interference with projection of afferent activity from the bladder to subcortical and cortical centers is delayed or deficient. Because the afferent information never reaches the cortical centers or reaches them too late, it does not provoke an appropriate response. Inhibition of detrusor activity is faulty and the detrusor reflex begins long before the patient is aware that his bladder is about to contract. It may be that there is also some interference with the descending inhibitory pathway which functions at a subconscious level because typically the bladder contracts at a lower volume than normal, although this is not invariably the case. Many diabetic patients, for instance, demonstrate unstable detrusor activity generated by bladder filling, but require a large urinary volume to provoke the response. In such patients, afferent pathways which provoke cerebral recognition and reflex responses appear to be deficient. Nevertheless, with overfilling, the reflex center does fire, but does so without the patient's foreknowledge or acquiescence. There is some evidence that within the spinal cord and peripherally, the sensory pathways which subserve cerebral

recognition of bladder events and, therefore, the volitional imposition of volitional control are disparate from those fiber tracts subserving only reflex activation of the detrusor.

In addition to overt supraspinal neural lesions, a number of other conditions may be associated with hyperreflexic but coordinate nonvolitional detrusor activity. Obstruction of the bladder outlet associated with compensatory hypertrophy is also associated with detrusor instability. In these patients, obstruction which develops gradually is accompanied by a compensatory change within the detrusor musculature. This results in low volume detrusor activity of high pressure and short duration. This kind of detrusor instability usually ceases either immediately or gradually after satisfactory reduction of outlet resistance by prostatectomy. In women with detrusor hyperreflexia, pronounced bladder muscular hypertrophy is not seen and bladder outlet obstruction is unusual. The most common cause of obstructive uropathy among women is massive cystocele formation rather than increased outlet resistance. Women with pronounced degrees of genital prolapse and retention generally have a thin-walled, decompensated bladder.[10] Symptoms of detrusor compensation with obstruction—urgency, frequency, incontinence—are very similar to those provoked by neural lesions. In both cases, the bladder may be trabeculated, and, among men, endoscopic evaluation may suggest obstructive prostatism. If a neurogenic condition is misdiagnosed as obstructive, and they often are, prostatectomy may be performed only to exacerbate the symptoms. The differential diagnosis can best be established by voiding cystourethrography which, in neurogenic conditions, will demonstrate a wide open proximal urethra and, in prostatism, occlusion of the proximal urethra at the time of a detrusor contraction.

Detrusor-external sphincter dyssynergia has been implicated in voiding dysfunction in women. This diagnosis is difficult to prove. A "guarding reflex" appears to be activated by a bolus of urine entering the urethra whereupon afferent pudendal fibers are excited and reflex contraction of the external sphincter occurs. This reflex does not require volitional or cerebral acquiescence and appears to be spinal. Activation of this reflex by a bolus of urine entering the urethra may be mistaken for detrusor-sphincter dyssynergia. Moreover, the common practice of reducing urethral outlet resistance in women by dilatation, internal urethrotomy, or, in some extreme cases, unilateral or bilateral pudendal blockage, seems

unwarranted in the light of evidence indicating that most of these patients do not have an actual increase in urethral closing pressure with the onset of a detrusor contraction.

In general, the treatment of detrusor instability associated with supraspinal lesions is disappointing. This is also true of a significant percentage of patients who demonstrate detrusor hyperreflexia without an overt neural lesion. The treatment is suppression of detrusor reflex activity by anticholinergic agents which may give the patient time to arrange to void in a socially acceptable manner. However, in many instances, anticholinergic agents also impair the patient's ability to generate a detrusor contraction volitionally and do little to improve the subjective warning time of reflex detrusor responses. Because the provocative element in the syndrome is bladder filling, if a patient cannot void volitionally "by the clock" and thus avoid a reflex detrusor contraction, then detrusor instability is likely to be as bad after treatment as it was before. The one thing that works well in ambulatory patients, in this circumstance, is a rigid program of timed voiding, say every 2 or 3 hours, together with the institution of anticholinergic therapy. Anticholinergic agents include imipramine hydrochloride (Tofranil), oxybutinin chloride (Ditropan), and probanthine (Probantheline Bromide). Treatment of detrusor instability with anticholinergic agents occasionally is totally unrewarding. Such patients are usually treated in desperation by indwelling Foley catheterization. If detrusor hyperreflexia is quite severe and very low volume bladder activity occurs, this may be unsuccessful. If the patient's bladder senses the catheter as a bolus of urine, it may try to expel it. With the onset of detrusor activity, the bladder neck opens widely and residual urine held around the Foley balloon but out of the reach of the eyelet of the catheter may be voided. Therefore, one may have the extremely unpleasant experience of placing a Foley catheter for incontinence only to find that the incontinence is as bad or worse with a Foley in place as before.

In addition to detrusor hyperirritability and neural lesions which influence directly the innervation of the bladder and urethra, a number of conditions should be ruled out before a patient is confined to Foley catheter drainage for incontinence. One of these is stress incontinence. In this condition, poor urethral and bladder base support are associated with hypermobility of the proximal urethra into the potential space of the vagina,[11] with an increase in intraabdominal pressure. Normally, the

proximal urethra is an intra-abdominal organ, closed by the involuntary activity of smooth musculature. An increase in intraabdominal pressure, because the proximal urethra is also intraabdominal, is reflected in both intravesical and intraurethral pressure and there is no net change in urethral closing pressure. If a deficiency of urethral supporting structures exists, an increase in intra-abdominal pressure forces the urethra through the defect in the pelvic floor into the vagina and a brief inequality of transmission of intra-abdominal pressure to the bladder and urethra occurs with consequent urinary spillage. The diagnosis of hypermobility-related urethral incontinence can be made on upright straining and resting cystograms. If the urethra moves inferiorly and posteriorly and is associated with urinary spillage, stress incontinence is present. Operative procedures to resuspend the urethra within the pressure influence of the abdominal cavity cure this kind of incontinence. If stress urinary incontinence is suspected but no urethral motion is seen on a straining radiograph, any operative procedure is unwise. These patients may be improved by agents with alpha stimulating properties and, in elderly women, topical estrogens may give dramatic relief.

Postprostatectomy incontinence can be diagnosed with reasonable accuracy using an upright cystogram. The bladder neck and prostatic urethra down to the area of the maximal skeletal muscle influence are patulous. A nonretention catheter perfused at a slow drip rate with a 20% Hypaque® and withdrawn from the bladder through the urethra will demonstrate the lesion on fluoroscopy. Egress of contrast material out of the side holes of the profile catheter enables identification of the point at which urethral closing pressure exceeds intravesical pressure (the level of continence). If the urethra at the level of the verumontanum is nonfunctional, the incontinence is probably related to the prostatectomy. A trial of alpha adrenergic stimulating agents (phenylisopropanolamine) is useful in such cases.

What type of evaluation is practical and useful in incontinent patients? Most patients, when questioned closely, accurately describe hyperreflexic detrusor response instability. Typically, they view this as an abrupt, detrusor response with absolutely no warning. Patients with stress incontinence complain of urinary loss related to an increase in intraabdominal pressure that typically occurs only in the upright or sitting position and not when supine. Isolated nocturnal incontinence is often due to a hyperreflexic detrusor response but occasionally is associated with a reversal of

the normal diurnal pattern. Elderly patients frequently produce more urine at night than during the daytime. This is particularly true of patients with mild congestive heart failure or cerebral vascular disease. Limiting fluid intake among patients with nocturnal incontinence on this basis is unrewarding, particularly among those patients receiving diuretic therapy at night or patients with fluid retention during the day due to inadequate cardiac output who mobilize interstitial fluid at night when cardiac activity is more efficient, regardless of the amounts ingested.

The diagnosis of detrusor instability can be made by cystometrogram. During initial filling, the bladder tolerates increasing volume with no great change in pressure. Suddenly, there is a brisk rise in intravesical pressure, at which time the patient notes that his bladder is contracting or about to do so. Normally, detrusor activity is not identified during a cystometrogram, and detrusor activity elicited simply by bladder filling is abnormal.

Most of the clinical problems of incontinent elderly patients can be managed without Foley catheterization if some attention is paid to the pattern of dysfunction in relation to the underlying disease process in a given patient. This, together with the use of urodynamic evaluation to answer specific questions, often produces information which provides effective rational management of the problem.

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